FEDERAL/PROVINCIAL
RESEARCH AND MONITORING
COORDINATING COMMITTEE (RMCC)

ASSESSMENT OF THE STATE OF KNOWLEDGE ON THE LONG-RANGE TRANSPORT OF AIR POLLUTANTS AND ACID DEPOSITION

PART 5

HUMAN HEALTH EFFECTS

AUGUST 1986

ACID RAIN
PLUIES ACIDES

Province of British Columbia
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Canada

ERRATA

Assessment of the State of Knowledge on the Long-Range Transport of Air Pollutants and Acid Deposition

Part 5, Human Health Effects

Page 5-5, paragraph 1

The third sentence should read as follows:

The incidence of colds going to the chest (38.3% vs 33.3%) and inhalant allergies (12.6% vs 5.1%) as identified from a questionnaire; cough with phlegm (10.0% vs 8.2%) and stuffy nose 25.3% vs 21.6%) as identified from a health diary were also higher in Tillsonburg than in Portage.

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PART 5
HUMAN HEALTH EFFECTS

ENVIRONMENTAL HEALTH DIRECTORATE
HEALTH AND WELFARE CANADA

AUGUST 1986

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HUMAN HEALTH

5.1. Introduction

Although acid deposition is most commonly known as "acid rain", the term actually encompasses both the wet and dry deposition of acidic substances. Acid deposition may take the form of rain, snow, hail or other "wet" precipitation. Alternately it may be deposited as dust, impacted fine particulate aerosols or by adsorption and absorption of gas. A second class of pollutants, referred to as "oxidants", is often associated with acid deposition because the same environmental condition can lead to the formation and transport of both types of pollutants.

Sulphur and nitrogen oxides are the primary sources of acid deposition. These oxides are released during the smelting of non-ferrous metals and during the combustion of sulphur-containing fossil fuels. Once released into the atmosphere, these primary pollutants may be transported by prevailing wind patterns over distances as short as a few kilometers or as long as several thousand kilometers, during which time they undergo complex chemical transformations. The resulting pollutant mix, frequently referred to as LRTAP (long range transport of air pollutants), consists both of the primary oxides of sulphur and nitrogen and their secondary transformation products including sulphuric acid and nitric acid aerosols, ozone, and small particulate matter--a large proportion of which falls in the respirable range (less than 10 microns (PM 10) in aerodynamic diameter.

Human health may be affected by contact with the air pollutant mixture (direct effects) or by contact with media affected by the pollutants, such as food or drinking water.

5.2. What are the direct health effects of long range transport?

5.2.1. Types of Studies

LRTAP airborne pollutants are generally divided into three major categories: sulphur oxide/SO_x suspended particulate complex; nitrogen dioxide/NO_x mixture (including nitrogen oxide, nitric acid, nitric aerosols and possibly nitrosamines) and photochemical oxidants, the principal component of which is ozone (O_3). Evidence for an association of LRTAP with direct adverse effects on human health is drawn from three sources: epidemiologic studies of exposed human populations, human volunteer studies (controlled chamber studies) and animal experiments. Each source and evidence has particular strengths and weaknesses.

Epidemiological studies can investigate the effects of real-life exposures in various population subgroups. It is difficult, however, to attribute observed adverse health effects to specific concentrations of any particular pollutant, to quantify dose-response relationships, or to exclude the influence of confounding variables that may contribute to the observed responses.

Experimental studies on human volunteers can identify and quantify human responses to individual pollutants but these findings are usually based on short-term acute exposures and cannot be indicative of the nature and magnitude of response to long-term chronic exposure.

Animal experiments provide information on a broad range of toxicologic effects and give insights into biologic mechanisms of response, but results cannot be directly extrapolated to human exposures. Both animal and human experimental studies tend to use healthy subjects and may not be applicable to subgroups of "sensitive" individuals within the general population that may be more susceptible to the effects of acidic pollution because of pre-existing respiratory or cardiovascular disease. Moreover, laboratory investigations usually involve the study of a chemically specific pollutant whereas LRTAP is neither chemically nor physically distinct, but a complex mixture that varies both temporally and spatially with regard to particle size, distribution, oxidation state and chemical species.

Nonetheless, there is increasing convergence from a wide body of evidence encompassing these three different approaches that acidic deposition is harmful to human health.

5.2.2. **Range of health responses to air pollution**

Direct health responses to the inhalation of airborne pollutants have been termed either acute or chronic. Acute responses are those occurring immediately or within a few days after exposure. Chronic health responses may not present themselves until there have been months or years of continual exposure and usually appear as increased rates of health deterioration or increased incidence of disease in exposed populations. There is also a wide range in the severity of respiratory health responses. Mortality falls at the extreme end of the spectrum; several models have been developed to estimate excess human mortality attributable to LRTAP; these will be discussed later. Respiratory morbidity, or illness, spans a variety of diseases, the most serious of which are emphysema, chronic bronchitis and asthma--collectively referred to as chronic obstructive lung disease. Other clinical symptoms indicative of either upper or lower respiratory tract infections include chronic coughing, cough with phlegm, croup, substernal chest pains, pneumonia, chest colds, wheezing, and allergic responses to

inhalant particules such as dust, animal fur or mould. Diminished pulmonary function, as measured by decrements in ventilatory rates, increased airway resistance or increased reactivity of bronchial passages are more subtle indicators of respiratory morbidity and are often used as end points in both experimental and epidemiological investigators, especially in studies involving children. The long-term biological significance of these changes is not at present clearly understood; however, there appears to be a statistical association between diminished respiratory function in childhood and the development of chronic obstructive lung disease in middle age, especially among individuals exposed to other respiratory health risks such as cigarette smoke, recurrent infections and occupational exposures (Burrows et al., 1979, Speizer et al., 1979). Pathological changes in lung tissue including, but not limited to, cancer, alterations in biochemical or immunological indices and decreases in mucociliary clearance rates (the rate at which inhaled particles are removed from upper respiratory tract surfaces) are also indicative of respiratory morbidity. Other effects of air pollution may include the aggravation of existing disease (such as asthma); increased severity of naturally ocurring infections; accelerated ageing changes; and diminution in levels of lung function attained in adolescence due to respiratory illness in childhood.

5.2.3. **Epidemiological studies**

The majority of epidemiological studies investigating the relationship between human health and air pollution has focused on urban areas where pollution levels from local industrial sources predominate. Three studies conducted in Canada have excluded urban and local industrial pollution and have investigated the effects of LRTAP on respiratory health.

(a) Hospital admissions and sulphate levels.

Using air pollution data obtained from standard ambient monitoring networks (1974, 1976 to 1982) from southwestern Ontario and correlating levels with admissions to hospitals in the same region, Bates and Sizto (1983) have reported positive association between ambient levels of sulphate, ozone, temperature and hospital admissions for respiratory illnesses. This analysis focused on two time intervals - July/August and January/February. The strength of the association was not dependent on asthma attack rates since the correlation was still significant when asthma-related admissions were excluded from analysis. The relevance of these findings is two-fold: first, sulphate levels in this region of Canada are predominantly due to LRTAP; and second the results of this study suggest that adverse health effects may be produced at normally occurring levels of sulphates. Furthermore, it may be assumed that for every case requiring hospital admission, a larger

number visited a hospital emergency department or a family physician; probably still more used increased medications; and a much larger number suffered some restriction of activity. The economic costs, therefore, implied by one increased admission may be considerable.

(b) Acute Exposure to LRTAP and Pulmonary Changes

A 10 day investigation examining the acute health effects of transported air pollutants was performed by Health and Welfare Canada in the summer of 1983 (Raizenne, et al., 1986). Using several measures of pulmonary function and self-reported symptomatology, 52 campers, ages 8 to 16 years, attending a residential summer camp in Ontario were studied. Twenty-three of the subjects were diagnosed asthmatics. Equipment located at the camp site monitored air pollution levels continuously.

A time lag function for fine particulates ($PM_{2.5}$), average sulphates, maximum daily ozone and temperature was associated with a decrease in the lung function of children. Significant decrements in forced vital capacity (FVC) and forced expiratory volume in one second ($FEV_{1.0}$), along with peak expiratory flow rates (PEFR) were observed in both the asthmatic and non-asthmatic campers. The results suggest that the single pollutant metric of fine fraction particles ($PM_{2.5}$) has an adverse effect on the lung function of children. It is generally accepted that fine particle sulphates represent the transported pollutant most likely hazardous to human health (OTA, 1984); this, however, is the first study to document the effect in an acute study design.

(c) Chronic Exposure to LRTAP and Pulmonary Function Changes

Population studies may be either cross-sectional, longitudinal or prospective, or retrospective in design. Cross-sectional studies examine differences in disease outcome and exposure between populations at a given point in time. Longitudinal or prospective studies follow the disease outcome in individuals exposed to different levels of pollution over time, using repeated or continuous measurements. Retrospective studies identify disease outcomes in individuals and attempt to associate them with past exposure to pollutants or risk factor of interest.

A cross-sectional study design was used by Health and Welfare Canada to investigate the potential chronic health effects of exposure to LRTAP in 1400 school children aged 7 to 12 years. Approximately half of the children resided in Portage la Prairie, Manitoba, a low pollution community and the other half in Tillsonburg, southwestern Ontario, where levels of airborne acidic pollutants are among the highest in Canada. Both communities are located in rural areas where there are no significant local

emission sources. Health assessments were based on respiratory questionnaires and standard lung function tests. Air quality was monitored in the communities by HWC for SO_2, NO_2, respirable nitrates and respirable sulphates. After controlling for the confounding influences of parental smoking habits and socio-economic status, and adjusting for age, sex and height, children in Tillsonburg exhibited a small (2%) but statistically significant decrement in lung function as compared with those in Portage la Prairie. The incidence of persistent chest colds (15.4% vs 12%), inhalant allergies (12.6% vs 5.1%), cough with phlegm (25.3% vs 21.6%) and stuffy nose (10% vs 8.2%) were also higher in Tillsonburg than in Portage. Although differences in a number of other respiratory symptoms did not reach a statistical level of significance, all showed a trend consistent with the major findings. While levels of NO_x and total respirable particles differed little between the communities, Tillsonburg had overall elevated levels of SO_2, respirable nitrates and respirable sulphates.

These findings are suggestive, but not conclusive, as it is unclear whether observed health differences were due to transported air pollutants or to some other unknown characteristics that differed between the two communities and were not measured in this study. A larger study involving five communities in Saskatchewan (control area) and five in southwestern Ontario (exposed area) is currently underway to verify and expand these results.

(d) Other air pollution studies

Other recent epidemiological studies, while not directly assessing the relationship between acidic deposition and human health, provide support for the association between components of acidic deposition and adverse respiratory health responses. Van der Linde et al., (1981) has monitored two communities in the Netherlands with differing levels of air pollution for 15 years. At intervals of 3 years, 2000 subjects aged 15-54 years complete respiratory health questionnaires and perform lung function tests. After nine years of study, inhabitants of the moderately polluted community showed a greater decrease in pulmonary function than did inhabitants of the rural community with low pollution levels. Furthermore, smokers compared with nonsmokers and heavy smokers compared with those who smoked less showed a greater decline in lung function. The prevalence of respiratory symptoms was also higher in the polluted region. Pollutants measured were SO_2, standard smoke (a surrogate indicator of total suspended particulate matter) and oxidants. Previous cross-sectional studies in the same population showed no systematic relationship between air pollution levels and lung function. This study is important in demonstrating the increased sensitivity of longitudinal studies in detecting health effects.

Two large U.S. studies (Ware et al., 1986) have reported associations between airborne pollutants and health responses. Of particular interest are the effects of photochemical oxidants demonstrated in the Los Angeles studies and the effects of fossil-fuel pollutants in the Harvard Six City Study. Longitudinal analyses of data from the Harvard Study indicates that exposure to moderately elevated concentrations of fossil fuel air pollution may increase the risk of developing chronic respiratory disorders. The fine fraction sulphate particulates ($PM_{2.5}$) are most strongly associated with adverse health effects.

5.2.4. **Experimental Studies**

a) Human Volunteers (chamber studies)

The importance of studies of human exposure lies in the ability to quantify the dose at which airway responses occur, to measure responses in hyperreactive persons, to study interactions between pollutants and to identify mechanisms of response. Ethical and practical consideration, however, limit human experiments to short term reversible effects and to adults without advanced stages of disease. Most experimental studies of human response to air pollutants have focused on measurements of ventilatory function or mucociliary clearance rates in normal or hyperreactive (including those with asthma or chronic bronchitis) subjects. Approximately 10% of the population can be medically identified as hyperreactive. Another 10-20% are believed to be clinically asymptomatic (i.e. they do not show medical symptoms associated with hyperreactivity) but exhibit hyperreactive responses in human volunteer experimental studies.

Ozone

Exposure to 0.5 ppm to 1 ppm ozone for 1 to 2 hours produces decreases in airway resistance, expiratory flow, vital capacity and diffusing capacity--all measures of pulmonary function (Young et al., 1964; Goldsmith and Nadel, 1967). Silverman (1979) exposed non-smoking asthmatics to 2 hours of 0.25 ppm O_3; although there was wide variability in responsiveness, approximately one-third of the subjects showed significantly decreased ventilatory function. Ozone at 0.4 - 0.6 ppm with exercise produces evidence of airway inflammation and increases in airway reactivity (Seltzar, et al, 1986). Although normal subjects vary in their sensitivity to ozone, the results on individual subjects are highly reproducible.

Nitrogen Oxides

The effects of experimental exposure to NO_2 have been investigated by Von Nieding and associates (1973) who found significant increases in airway resistance of healthy subjects after 1- to 15-

minutes exposures to NO_2 (1.6-2.5 ppm). Orehek et al., (1976) evaluated the interaction between exposures to NO_2 and bronchoconstrictal agents in subjects with asthma. NO_2 concentrations of 0.11 ppm alone caused a significant increase in airway resistance in 3 of 20 subjects. The same NO_2 concentrations, however, significantly increased the bronchial sensitivity to the bronchoconstricting agent, carbachol, in 13 of the 20 subjects. In the remaining 7 subjects NO_2 alone or in the presence of carbachol had no effect. It is not known whether similar ambient concentrations of NO_2 will enhance the effect of naturally occurring bronchoconstricting agents.

Sulphur dioxide/sulphate particulate mix

There appears to be wide variability of response among individual human subjects to SO_2. Some apparently normal persons in the studies of Nadel et al., (1965) and Lawther et al., 1974a,b,c) showed marked airway reactivity at the low range of doses. Increased airway resistance (constriction of passages) has been demonstrated in healthy persons experimentally exposed to SO_2 at concentrations of 5 ppm or more and hyperreactive individuals may respond to short-term exposures of less than 1 ppm (Nadel et al., 1965; Wein and Bromberg, 1973).

In the atmosphere SO_2 is converted to sulphate particles. Two main types of sulphate particles: sulphuric acid (H_2SO_4) and ammonium sulphate and bisulphates are extremely small and may be inhaled deep into the lung. The high acidity of sulphuric acid particles make them of primary concern to public health, although ammonia in human breath may neutralize sulphuric acid. Instruments generally used by networks for monitoring sulphates do not distinguish between the different chemical species, some of which are more biologically active than others. A procedure for measuring sulphate speciation in ambient air has been recently been developed by Allen et al., (1984); this method, however, is not yet in widespread use.

In chamber experiments, acute exposures to high concentrations of sulphuric acid aerosols have a variety of adverse health effects. Concentrations of about 750 ug/m^3 have irritated eyes and temporarily decreased vision; concentrations of 350 ug/m^3 have increased breathing rates and altered lung function in asthmatics. Changes in lung clearance rates have been observed in healthy non-smokers at concentrations of 100 ug/m^3.

There also appears to be a "carrier" effect of fine air particles (Koenig et al., 1982) whereby toxic gases or metallic ions may absorb or attach to the particles and penetrate deep into the respiratory tract, bypassing natural defense mechanisms. This

phenomenon has not been well studied although there is evidence suggesting that the "carrier" effect increases under conditions of higher humidity (McJilton et al., 1976).

b) Animal Studies

Environmental studies on animals can establish the biologic plausibility of an association between specific pollutants and adverse human health and can elaborate the physiological and biochemical pathways for these responses. The animal literature will not be extensively reviewed here but only summarized briefly.

Sulphur dioxide/sulphates

Adult male beagle dogs were exposed to high concentration of SO_2 (500-600 ppm) in a gas chamber for 2-hour periods twice a week for 4 to 5 months (Asmundson et al., 1973). Exposure resulted in hyperplasia of the bronchial glands and an excess of mucopurulent exudate in the bronchial tree. Exposure of rats and hamsters (Asmundson et al., 1973) to concentrations of SO_2 (100-400 ppm) produced variant effects ranging from the loss of cilia, vacuolation, pyknosis and extrusion of cells after 1 to 2 days of exposure at 100-200 ppm. At higher doses (400 ppm) squamous metaplasia was produced after 1 to 2 weeks. Sulphur in the form of sulphuric acid or sulphate aerosols produced a greater broncho-constrictive effect than did equimolar concentrations of SO_2 (Amdur, 1973).

The most sensitive animal in respiratory function tests after short term exposure to sulphuric acid was the guinea pig; airflow resistance and decreased compliance resulted after exposure to 100 ug/m^3 sulphuric acid, 500 um/g^3 ammonium sulphate, and 900 ug/m^3 ammonium bisulphate (Amdur et al., 1978). Other studies gave equivocal results on donkeys, dogs, rats, and guinea pigs at concentrations from 800 to 100,000 ug/m^3 sulphuric acid and 400 to 2100 ug/m^3 ammonium sulphate. (U.S. EPA, 1982).

Clearance mechanisms appeared to be more generally affected than respiratory functions. A concentration as low as 200 ug/m^3 sulphuric acid for 1 hour caused decreased tracheobronchial clearance in donkeys (Schlesinger et al., 1978). It has been suggested that variable amounts of ammonia both in the exposure chambers and in the subjects' respiratory tracts could explain some of the variability or species responses; ammonia could have neutralized part of the acid (Schlesinger et al., 1978).

Ozone

Ozone produces morphological and biochemical alterations in experimentally exposed animals: the overall changes evoked by exposure

are generally the same for different species but vary with the dose and time course between exposure and response. Pulmonary edema, an acute response to O_3 exposure, may be detected after six hours of exposure at concentrations of approximately 0.25-0.5 ppm in the rat (Alpert et al., 1971). Increased susceptibility to airborne infectious agents occurs in rodents with exposure to concentrations of ozone as low as 0.08 ppm for 3 hours (Coffin et al., 1968); these data suggest that immune function is compromised in the presence of low doses of the oxidant. Hepatic effects are detected in mice (Gardner et al., 1974) and could have broad implications for overall metabolism of foreign compounds by exposed populations but insufficient data at this time are available for extrapolation to human health effects.

Ozone has recently been shown to increase the incidence of lung tumours in mice chronically exposed to 0.34 and 0.51 ppm of ozone over a period of 6 months (Hassett et al., 1985) and thus may be carcinogenic in animals. However, these experiments utilized a lung tumor-sensitive species of mice and must be replicated in other animals using standard NCI protocol before conclusions can be made regarding carcinogenicity of ozone.

Nitrogen dioxide

Morphologic studies of animals exposed continuously to concentrations as low as 0.12 ppm NO_2 have revealed distinct alterations in the alveoli and respiratory bronchioles (Freeman et al., 1968). Mice exposed to 0.5 ppm for 12 months had similar morphological alterations as those observed at much higher concentrations; although clear-cut dose-response data are not available, the concentration of NO_2 seems more important than time of exposure in determining response. Other experiments (Gardner et al., 1978) also suggest that short-term exposures may be more toxic than were previously supposed. As with ozone, increased infectivity produced by infectious agents occurs in several rodent species (Hadley et al., 1977), and hepatic damage occurs with long-term exposure to concentrations of 0.5 ppm (Minzet et al., 1977).

5.2.5. **Assessing the Overall Health Risks of Long Range Transport**

Evaluation of health risk involves four component tasks (Lowrence, 1976): (1) identifying the health effects of long range transport (2) quantifying these effects at various concentrations (3) estimating how many people are exposed at these concentrations and (4) calculating the overall health risk associated with a given level of acid deposition.

The preceeding sections have documented a large body of evidence concerning the adverse health effects of constituent pollutants of

long range transport. Although levels of aerosol sulphates, respirable particles, and ozone currently occurring in the Canadian environment are probably associated with increased respiratory morbidity, the extent and magnitude of these effects have not yet been precisely measured. This is due in large part to the inherent difficulty in distinguishing between pollutants that are closely interrelated. Health responses to these pollutants may also span a wide range in terms of magnitude of effect; they can be acute, chronic, delayed or cumulative and it is difficult to isolate the effects of LRTAP from other contributing factors.

Quantifying LRTAP-related health effects at various concentrations involves the formualtion of dose-response curves as modified by age, susceptibility and other sociodemographic and environmental variables, and the application of each set of damage functions for a pollutant to the dose estimates for all segments of the population. Because of the paucity of data such risk estimates are both uncertain and judgemental, due in part to possible errors in the measurements of exposure levels and health effects, and to the necessity of making assumptions that can be neither proved nor disproved scientifically. However, similar processes of risk estimation occur in many other areas of public policy such as pesticide registration, energy conservation and evaluation of potentially carcinogenic substances in the environment.

To date, little work has been done to quantify the total health risk of airborne acidic deposition in Canada. Hamilton's model, although widely quoted, yeilds estimates of excess mortality that are too uncertain to be useful. In addition, a number of the model's assumptions regarding exposure estimates are based on limited U.S. data and are not applicable to the Canadian environment. Clearly, more research in this area is needed.

Estimating mortality attributable to LRTAP does not necessarily imply that mortality impacts are the most significant. Mortality dose functions have been used because the available data on morbidity are inadequate for detailed analyses. A recent preliminary analysis by the DPA Group and Associates (1984) for Environment Canada calculated morbidity impacts due to LRTAP by estimating respiratory morbidity as a function of excess mortality. While not distinguishing among disease symptoms, they estimated that respiratory disease due to LRTAP incurred an annual average of 152,800 disability days, including 42,500 work days, 49,900 doctor visits and 185,500 hospital days. As with mortality, these estimates exhibit a wide range of uncertainty.

5.2.6. **Summary**

National primary air quality standards in the U.S. and Canada are presented in Table 1. There is ample evidence that all of these

"criteria" pollutants (those for which national air quality standards exist) produce toxic effects in animals and adverse health effects in humans exposed to sufficiently high concentrations. Major differences in judgement exist within the scientific community as to the appropriate margin of safety to be applied to primary air quality standards and whether current standards provide a large or small margin of safety. Toxic effects have been observed in apparently healthy laboratory animals exposed to concentrations that are 2- to 10- fold greater than ambient concentrations or are even sometimes reached in ambient air (Shy et al., 1979).

Historically, epidemiological studies associating air pollution with an increase in adverse health responses were never intended for use in setting air quality standards since methodologies did not provide accurate estimates of either dose or response. These studies relied primarily on measurement of ambient pollutant concentrations as an estimate of human exposure. The main advantages to utilizing air quality network monitoring data to estimate human exposure to air pollutants were cost and convenience. However, air quality monitoring networks are operated primarily for purposes of compliance and abatement, and not for human exposure assessment. The sites are located outdoors, often some distance away from residential areas and in many cases close to an emission source such as a smelter. Studies investigating the representativeness of ambient air monitoring data with respect to actual human exposure (as measured by personal monitors) have found it to be an underestimate in general (Ott and Mage, 1974; Cortese and Spengler, 1976). Moreover, those components of air pollution that are most likely to impact on human health--fine particles and transformation products, are not routinely measured. Additionally, the use of ambient air data only to estimate human exposure does not consider contributions from other microenvironments (e.g. occupational, residential).

More sophisticated monitoring equipment measuring components of airborne pollution that are relevant to human health are in use in epidemiological studies on acid deposition currently being conducted by Health and Welfare Canada. New methods for better estimation of human exposure, utilizing a microenvironmental approach and personal dosimeters are also being presently developed and tested by Health and Welfare Canada. More accurate estimates of personal exposure are essential for the development and quantification of dose-response curves.

Several additional factors merit serious consideration in the evaluation of health risks associated with acidic deposition.

(a) Transformation products of long range transport

There are no national standards for the transformation products of SO_2 and NO_2, such as sulphate or nitrate-containing particulate matter, acid aerosols and fine particles. As previously mentioned, instruments presently used for monitoring ambient sulphates do not distinguish between different chemical species of sulphates, some of which are more biologically reactive than others. Even less is known about the human health effects of nitrate aerosols and their concentrations in the atmosphere.

Fine suspended particules [less than or equal to 2.5 microns ($-PM^{2.5}$)] comprise a large proportion of the particulate matter associated with acid deposition. Numerically, fine particules may constitute the bulk of particles in long range transport; yet by mass, they appear to contribute much less to total TSP because coarse particles are so much larger (Lundgren and Paulis, 1975). Present air quality standards do not distinguish among particles on the basis of size but apply only to TSP. Fine particles appear to have much greater consequences for human health. First, because of their size, fine particles may by-pass mucociliary clearance and other respiratory defense mechanisms in the upper respiratory tract and penetrate deep within the respiratory tract. Second, because of their large surface area in proportion to their mass, fine particles are efficient sites for the condensation of volatile gases in atmospheric pollutants, the absorption and concentration of pollutants and various secondary chemical reactions (Porera and Ahmed, 1979). The deposition of these particles deep in the lung may expose small areas of relatively unprotected lung tissue to much higher concentrations of toxic and/or carcinogenic pollutants than would occur in purely gaseous exposures (Nahishc, 1978; Page et al., 1985).

(b) Long range transport pollutant mix

Transformed products are potentially more toxic on a per-unit basis than their gaseous precursors because they aggregate, interact and exhibit unique toxicological characteristics. In some studies, additive or synergistic effects of two air pollutants have been observed in humans and animals (e.g. Bates and Hazucha, 1973). In others, the relative toxicity depends on the sequence in which subjects are exposed to more than one pollutant (Hassett et al., 1985). Generally, few experimental data are available for combinations of air pollutants and their effects on animals or humans, in part because the physical and chemical nature of the complex mix have not been well characterized and vary both temporally and spatially in ambient air. Thus, when experimental results are compared with the actual population exposures to a large number of varying long range transported pollutants, it must be concluded that sufficient data are not as yet available to assess the toxicity of this pollutant mix.

(c) Population at risk

Most of the existing data base on the health effects of acidic pollutants was developed from studies on healthy human subjects or animals. It is generally believed that air pollution plays an important role in the exacerbation of adverse health responses in susceptible persons and in the acceleration of the progression of chronic respiratory disease. The elderly, asthmatics, persons with pre-existing cardio-resiratory disease and children are considered to be at high risk to the adverse effects of acid pollution, because of either already compromised health status or, in the case of children, enhanced susceptibility during a period of rapid biological growth. Additionally, children may receive a much higher dose than adults breathing the same polluted air because they breathe more air per unit of body weight than adults and have far fewer air sacs or alveoli, the site in the lungs where gas exhange takes place (Phalen and Oklham, 1985). Further epidemiological and experimental research is required in order to evaluate more quantitatively the health responses of persons with compromised health and of children.

5.3 What are the indirect health effects of long range transport?

Acid deposition may affect human health indirectly through two routes: the bioaccumulation of chemicals in human food, particularly mercury in fish; and the contamination of drinking water supplies by increasing the levels of heavy metals such as lead, copper, zinc, cadmium, mercury and aluminum.

5.3.1. Bioaccumulation of Chemicals in Human Food

The detection of increased levels of trace contaminants in humans originating from food is complicated by existing background body levels in the population and the fact that the increasingly varied diet of Canadians comes from many geographic areas. However, there is major concern that mercury contamination in edible fish could pose a serious problem for public health.

Acid deposition appears to cause changes in the biogeochemic cycle of metals, principally mercury (Hg), resulting in increased bioaccumulation in fish. Elevated Hg levels have been found in fish inhabiting lakes far removed from local sources. These lakes were characterized by low alkalinity and thus were poorly buffered. The mechanisms by which acidification might increase the mercury content of fish are not fully understood. These processes are believed to be extremely complex and involve redox reactions, inorganic and organic sequestering agents and the nature of the particular food chain in question. It is felt that although low pH does not necessarily increase the absolute water content of Hg, it does increase the bioavailability of Hg.

It has been observed that fishes from low pH lakes have elevated mercury concentrations, in some areas. The species and trophic level of fish are additional contributing factors. Some larger piscivorous fish have been observed to have a higher mercury content than planktivores. These fish are usually the most prized sport fish and therefore make up the majority of the yearly catch consumed by recreational sportspersons.

It appears that natural sources contribute the major portion of mercury to the aquatic environment, but direct atmospheric depositions can be significant on a regional scale. Air samples taken downwind from a major coal-fired generating station in Tennessee were analyzed (Lindberg, 1980). It was found that mercury was emitted predominantly in the elemental vapour phase with little conversion to particles. This finding supports the theory that mercury discharged from a point source is not deposited locally, but rather may be subject to transport on a regional scale. Since Hg is in the vapour phase, it is believed that the major mechanism for atmospheric removal of Hg is via precipitation scavenging. It is also felt that this process should increase in efficiency as the acidity of the precipitation increases.

The toxicity of methylmercury in humans is well described in the literature following catastrophic poisonings from consumption of contaminated fish in Japan in the 1950's and '60's and of treated seed in Iraq in the winter of 1971-72 (Chang, 1977). Studies on a number of cases of industrial poisoning have also contributed substantially to the understanding of the toxic effects of methylmercury in the nervous system of man (WHO, 1976).

5.3.2. **Contamination of Drinking Water**

Since most drinking water in Canada is obtained locally, trace contaminants mobilized by acid deposition could be an important route of human exposure. Acid deposition may enhance the scavenging of atmospheric contaminants, increase the leaching of metal from watersheds and sediments, and via corrosion, mobilize natural or synthetic contaminants of materials used in water storage and distribution systems. All of these factors may degrade drinking water quality. It is therefore necessary to consider the nature of possible trace contaminants, the potential effect of acid deposition on those contaminants, the susceptibility of the various sources of drinking water to contamination and the size of the population at risk (McDonald, 1985).

5.3.2.1. **Sources of Drinking Water**

The major sources of drinking water are groundwater and surface water; the most important minor source is cistern water. The source

of drinking water is an important factor in determining its potential for being adversely affected by acid deposition (McDonald, 1985).

a) Groundwater. Certain shallow springs and wells in Eastern Canada may be affected by acid deposition. The highly buffered nature of most deep groundwater in Ontario and Quebec makes it unlikely to be affected by acid deposition. However, groundwater in the Atlantic Provinces is potentially at risk due to the poor buffering capacities of many Maritime soils. In Sweden and Germany, groundwater has been affected by acid deposition (Hubbard and Wenblad, 1980).

b) Surface waters in Canada, U.S. and Scandinavia have been damaged by acid deposition. Small, shallow surface water basins may be more highly susceptible than deeper streams and lakes, especially when runoff is taken into consideration.

c) Cisterns. Depending on their construction, cisterns may have little capability to buffer against acid deposition. For example, if a cistern is constructed with vinyl liner, no buffering of acid deposition will occur. Therefore, persons whose drinking water is primarily obtained from cisterns may be at the greatest risk of adverse health effects emanating from acid deposition. The number and distribution of drinking water cisterns in Canada is currently unknown.

5.3.2.2. **Environmental contamination of drinking water supplies**

Most contaminant mobilization studies are based on decreasing pH, and a substance's potential for increase is based primarily on the contaminants of concern and their ambient background levels. Recommended maximum acceptable concentration (MAC) in Canada, and U.S. EPA's maximal contaminant levels (MCL) are presented in Table 2.

a) Mercury. Methylmercury and associated short-chain alkyl mercurial compounds are dangerous environmental contaminants, primarily because they accumulate in edible fish tissue, as previously discussed. Most drinking water samples from North America and Europe contain low levels of mercury. However, levels above the 2 ug/L MCL set forth in the National Primary Drinking Water Regulations (NPDW) have been reported for 24% of rural U.S. households (Frances et al., 1984). Although the concentration of mercury in drinking water would not typically constitute a substantial portion of the maximum safe intake limit of 30 ug/day, occupants of rural households where mercury concentrations exceed the 2 ug/L may be at special risk from any increase in mercury in their drinking water due to acid deposition.

b) Aluminum is not known to be utilized in any natural biological process and in the past it has been considered to be relatively non-toxic to humans. It has recently become a health concern because of its increased mobilization in acid waters (Wright et al., 1980). Elevated levels of aluminum of up to 0.3 ppm have been reported in acidified lakes and streams in Eastern Canada. At normal pH, most aluminum is present in insoluble forms and thus is not biologically available. However, the complex chemistry of aluminum changes drastically as the pH decreases and more aluminum becomes solubilized, producing high concentrations in affected watersheds. Moreover, aluminum is widely used as a coagulant in the treatment of drinking water: alum-treated water shows aluminum levels on average three times greater than those of untreated water (Miller et al., 1984).

Within the past several years, aluminum has been implicated in the pathogenesis of several human disorders. At relatively low levels, aluminum has been implicated in dialysis dementia, a disorder of the central nervous system. Aluminum poisoning is recognized in patients with renal disease (Wills and Savoy, 1983). High aluminum levels have been linked with Alzheimer's Disease (Shore and Wyatt, 1983), as well as with other disorders of the central nervous system, such as amyotrophic lateral sclerosis and dementia complex of Guam; however, its role in the etiology of these degenerative diseases remains unknown.

According to a study by Shore and Wyatt (1983), the human diet averages approximately 22 mg/day of aluminum. The maximum level of aluminum found in Miller et al.'s (1984) study of 200 drinking water systems in the U.S. was 2.7 mg/L. Assuming a water intake of 2 L/d, the maximum daily human intake of aluminum from water would be 5.4 mg/d. This amount is less than 25% of the mean total daily ingestion and considerably lower than the 35-108 mg of aluminum found in antacids and buffered aspirins, which are frequently ingested in multiple daily doses over prolonged periods.

Thus, the potential for toxicity to humans caused by increased aluminum levels in drinking water appears low for healthy adults. While there are no drinking water guidelines for aluminum currently available in Canada or the U.S., consideration should be given to the development of guidelines, given the potential role of this metal in dialysis dementia and several degenerative diseases of the central nervous system.

5.3.2.3. **Contamintion Due to Corrosion Potential in Water Distribution Systems**

The corrosion potential or aggressiveness of drinking water can be increased by a decrease in pH occurring as a result of acid deposition. Generally, in large urban drinking water treatment facilities, this problem is overcome by adjusting the pH of the water

before distribution. However, in smaller, less-regulated facilities, particularly as occur in rural areas, an increase in aggressiveness may expose humans to toxic concentrations of metals and asbestos from drinking water distribution and storage systems and household plumbing (Millette et al., 1980). Corrosion products of primary concern to human health are copper, lead, cadmium and asbestos.

a) Copper is a necessary trace element in the human diet; human intake of copper has been estimated at 2-5 mg/day. Copper is often found in surface and groundwater at low levels. However, a rise in copper levels in soft, low pH drinking water has been documented (Schroeder et al., 1966) and has the potential to create a health hazard. Copper is widely used in potable water service lines; as the pH of drinking water in copper piping decreases, there is an exponential increase in both standing and flowing water. The MAC and MCL for copper is 1 mg/L (Table 2). Copper levels can reach 78 mg/L for short periods after corrosive water stands in pipes and taps overnight, but decline rapidly after the first flush of running water. Copper levels greater than approximately 5 mg/L are usually unpalatable and induce vomiting at levels greater than 15 mg/L. Thus, the potential for damage caused by increases in copper levels in drinking water impacted by acid deposition appears small and self-limiting.

b) Lead There is a natural background level of lead in water, but the average level in water supply systems is higher than background. Most major centres no longer use lead as a service line material for new installations, but a large amount is still in place in older water systems. Lead levels in older water systems average 30 ug/L and are 3 times higher in corrosive drinking waters in first-flush samples than from running tap water (Moore et al., 1977).

Lead is a highly toxic metal that is widely distributed in the environment. Pregnant women and children are most at risk from lead contamination of drinking water (Richards and Moore, 1984). Because lead can cross the placenta, fetuses can absorb up to 50% of ingested lead; the corresponding amount for adults is approximately 8%. Absorbed lead also penetrates the blood-brain barrier more rapidly in fetuses, infants and children than in adults. High blood lead levels in children (7.30 ug/mL) induce biochemical and neurophysiological dysfunction (Clarkson et al., 1983). Low blood levels of lead have been implicated in IQ deficiencies and behavioural problems. Consumption of drinking water with first flush lead levels of 800 ug/L or more by females during pregnancy has been correlated with increased mental retardation in their infants.

Lead levels as high as 3 ug/L have been found in areas with lead plumbing and acidified water (pH 5.5). Therefore, acid deposition may increase the overall human health risk by adding to the body

burden; at present, this effect cannot be quantitatively estimated.

c) Cadmium Although food is the major source of cadmium for the general human population, cadmium is acid soluble and its solubility increases exponentially with decreasing pH. Cadmium can solubilize in drinking water through corrosion of galvanized pipe or of copper-zinc solder used in distribution systems (deBarry et al., 1982). The major adverse human health effect associated with long-term low level ingestion of cadmium is renal disease. The biological half-life of cadmium is approximately 30 years; thus, nearly all cadmium ingested over a lifetime is retained. Therefore, from a public health perspective, this metal is a contaminant of serious concern in drinking water.

Elevated cadmium levels have been recorded in both rainfall (Aben et al., 1983) and drinking water supplies impacted by acid deposition (Meranger et al., 1983; Young and Sharpe, 1984). Subsistence diets or dietary deficiencies also exacerbate damage to health from cadmium. However, simultaneous ingestion of increasing levels of copper, iron or zinc (which counteract cadmium's adverse health effects) may mitigate the damage caused by cadmium. All these metals often co-occur in acidified water.

Several field studies have documented the acidity and metal concentration in drinking water in rural areas in both the United States and Canada. HWC conducted studies during the summer of 1981 (Meranger et al., 1983). The sites chosen for the operation were cottages in central Ontario, where acid deposition rates were moderately high. Metal concentrations were measured in the raw water and at the tap, following passage through the plumbing system. The leaching rates of metals from the piping system were also calculated for copper, lead, zinc and cadmium.

While the metal concentrations of the raw water were slightly elevated above background, maximum values of 4560 ug/L copper, 478 ug/L lead, 3610 ug/L zinc and 1.2 ug/L cadmium were recorded from tapwater. These readings were obtained after the water had been standing in the lines for 10 days. The concentrations of all metals decreased after flushing, but remained above source levels, indicating that the metal concentrations were related to residence time in the pipes. The results from the leaching study indicated that the water in the Muskoka-Haliburton region of Ontario had a maximum leaching rate which occurred during the first two hours that the water was in the system. It was also found that metal levels in the water continued to rise for up to ten days.

The conclusions of this study were that although the metal levels may periodically exceed acceptable levels, flushing the distribution system daily will reduce the metal levels and the potential risk of developing health problems.

Fuhs and Olsen (1979) and Young and Sharpe (1984) investigated drinking water samples in the Adirondack region of the U.S.A. The results of their sampling showed that the pH of the water was depressed and that there were elevated levels of copper and lead. One home had copper and lead values of 6600 and 100 ug/L respectively, while another had a copper concentration of 2300 ug/L. Both of these homes derived their water supply from shallow wells located near the house. In fact, 11% of the homes with wells had lead levels in excess of 50 ug/L. In 1980, 22% of the homes with cisterns had mean standing water Pb concentrations above 50 ug/L. One of the conclusions of this study was that high concentrations of metals may be found in homes in this region with metal piping or cisterns, especially if the lines are used intermittently.

d) Asbestos In some areas in the U.S. and Canada, asbestos- cement (A/C) roofing tiles have been used for many years as catchments for household drinking water cisterns. A/C piping is used widely in U.S. water distribution systems; according to deBarry et al. (1982), 320,000 km of A/C piping is now in service and accounts for one-third of current sales. Similar data on the use of A/C piping are not available in Canada.

Airborne asbestos is a potent human carcinogen. Although the risk of cancer associated with asbestos contamination of drinking water is not well documented, a suggested MCL of 300,000 fibres/L has been announced by EPA. No Canadian guidelines are available. Recent evidence suggests that aggressive drinking water can corrode A/C materials and leach large amounts of fibres into the water (up to 500 x 10^6/L) (Millette et al., 1980). Between 10 and 40 million people in the U.S. may be ingesting asbestos in their drinking water. The number of persons similarly exposed in Canada is currently unknown.

5.3.2.4. **Population at Risk**

The population most at risk from contaminated drinking water supplies appears to be those using small, private or rural facilities in acid-sensitive regions (Clarkson et al., 1983; Frances et al., 1984). Operators of these facilities usually do not monitor water quality routinely and provide, at best, rudimentary treatment.

The size of the rural population in Canada living in acid-sensitive areas and obtaining drinking water from small, private or rural facilities is currently unknown. Crude estimates of the affected U.S. population have been made (McDonald, 1985) using U.S. 1980 Census of Housing data and data obtained from EPA's National Statistical Assessment of Rural Drinking Water Conditions. Estimates were made of the number of housing units in acid- sensitive northeastern states using dug wells and "other sources", primarily cisterns, for their drinking water. These sources are believed to be most at risk to acid deposition. An estimated 601,175 housing units in this

region, with an estimated median value of 2.65 occupants per rural household, met these criteria, for a total of 1,592,849 persons. Based on the results of a nationwide survey of 2654 rural households, the percentages of individuals exposed to levels of lead, cadmium and mercury exceeding the maximal contaminant levels established by EPA were calculated. McDonald applied these same percentages to those persons with susceptible water supply systems in the northeast and estimated that there may be between 25,000 - 350,000 persons who drink highly contaminated water.

5.3.3 Summary

Acid deposition poses the potential for causing damage to human health through increases in the levels of contaminants in drinking water. Contaminants that merit further research because of their toxicity and potential concentrations in acidified drinking water are corrosion products, such as lead, cadmium and asbestos, and environmentally-occurring mercury and aluminum. Although Nova Scotia has lower rates of acid deposition than Ontario and Quebec, the poor buffering capacity of soils and bedrock render surface and groundwater in this province highly susceptible to acidification. Future research must address this issue.

Research is also needed to document the type and distribution of drinking water sources in rural areas in Canada impacted by acid deposition. Estimates of affected populations and of existing levels of the various contaminants in drinking water to which the affected populations are exposed must be developed. The effects of human health caused by sudden addition of contaminants from episodes of snowmelt and spring runoff should also be ascertained.

As with airborne acidic pollutants, subpopulations at high risk to the adverse health effects of contaminants in acidified drinking water need to be identified.

It must also be remembered that the health of people living in acid-deposition affected areas may be at even greater risk because of the joint contributions of airborne acid pollutants and drinking water contaminants, whose concentrations are increased by acid deposition.

It is essential that educational programs be developed and implemented so that populations at risk can take mitigative steps to reduced their exposures, if necessary.

Table 1. Current Health-Related National Ambient Air Quality Objectives

Pollutant	Canada (maximum acceptable level)		United States	
	Level	Time	Level	Time
Ozone (O_3)	0.08 ppm 0.25 ppm 0.015 ppm	1 hr 24 hr Annual[b]	0.12 ppm (235 ug/m^3[a])	1 hr
Nitrogen oxides (NO_2)	0.21 ppm 0.11 ppm 0.05 ppm	1 hr 24 hr Annual[b]	0.05 ppm (100 ug/m^3)	Annual[b]
Sulphur oxides (SO_2)	0.34 ppm 0.11 ppm 0.02 ppm	1 hr 24 hr Annual[d]	0.14 ppm (365 ug/m^3) 0.03 ppm (80 ug/m^3)	24h[c] Annual
Suspended particulate matter	120 ug/m^3 70 ug/m^3	24 hr Annual	260 ug/m^3 75 ug/m^3[e]	24 hr[c] Annual[d]

[a] The standard is attained when the expected number of days per calendar year with maximum hourly average concentrations above 235 ug/m^3 is equal to or less than 1.
[b] Annual arithmetic mean.
[c] Not be exceeded more than once per year.
[d] Annual geometric mean.
[e] Currently under review and possible revision.

Table 2. Drinking Water Contaminants of Concern: Recommended Limits in Canada and the United States

Contaminant	Canada		United States	
	Maximum Acceptable Concentration	Background Level	Maximum Contaminant Level	Background Level
Aluminum	None	less than 1000 ug/L (in surface waters)	None	less than 0.03 ug/L
Arsenic	50 ug/L	less than 5 ug/L	100 ug/L	less than 100 ug/L
Cadmium	5 ug/L	less than 1.0 ug/L	10 ug/L	less than 1.0 ug/L
Copper	1000 ug/L	10 to 180 ug/L	1000 ug/L	less than 0.05 ug/L
Lead	50 ug/L	7.6 ug/L (average)	50 ug/L	1 to 10 ug/L
Mercury	1 ug/L	less than 0.1 ug/L (less than 0.3 in Atlantic provinces)	2 ug/L	less than 0.04 ug/L
Uranium	20 ug/L	less than 1.0 ug/L	None	less than 2.0 ug/L
Asbestos	None	10^5-10^6 fibers/L	300,000 fibers/L	10^4 -10^8 fibers/L

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